JACC Vol. 32, No. 1
July 1998:97–102

Time Dependence of Left Ventricular Recovery After Delayed Recanalization of an Occluded Infarct-Related Coronary Artery: Findings of a Pilot Study

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Objectives. **We sought to test the hypothesis that late recanalization of infarct-related coronary arteries (IRAs) improves long-term left ventricular (LV) function.**

Background. **Reperfusion within 24 h of an acute myocardial infarction (MI) has been shown to improve myocardial healing and to reduce infarct expansion. Uncontrolled data suggest that there may be a time window of several weeks for such an effect.**

Methods. **Sixteen asymptomatic patients 10 ± 4 days after a first Q wave anterior wall MI with persistent left anterior descending coronary artery occlusion and infarct-zone akinesia were randomized to immediate (2 weeks) or delayed (3 months) angioplasty. Repeat catheterization and cardiac magnetic resonance imaging (MRI) were performed after 3 and 12 months.**

Results. **Angiography 3 months after MI revealed that LV ejection fraction (LVEF) had increased ([mean ± SD] 54.4 ± 4.3% vs. 63.9 ± 7.4%, $p < 0.01$) as a result of improved regional function ($p < 0.01$) and LV end-systolic volume had decreased ($p < 0.002$), whereas LV end-diastolic volume remained unchanged. With delayed angioplasty, LVEF, infarct zone wall motion and LV volumes did not improve. Cardiac MRI at baseline and at 3 and 12 months confirmed these findings and extended them up to 1 year, indicating that delayed angioplasty could no longer improve LV function because of marked LV dilation ($p < 0.01$). Immediate angioplasty had a high success rate, but restenosis (50%) was accompanied by new severe angina as a clinical indicator of salvaged myocardium, which did not occur after delayed angioplasty.**

Conclusions. **This pilot study in selected patients supports the hypothesis that myocardial viability persists ("hibernation") for 2 to 3 weeks but not for 3 months after MI, during which time it may be worthwhile to restore blood flow to a large myocardial territory, even in asymptomatic patients, to improve long-term LV function.**

(J Am Coll Cardiol 1998;32:97–102)

The open artery theory (1–4), which holds that immediate reperfusion of an infarct-related coronary artery (IRA) results in myocardial salvage, preserved left ventricular (LV) function, and thus improved survival has recently been supported by several large clinical trials (5–8). It has further been hypothesized that delayed reperfusion of infarcted myocardium may also be beneficial (9) by improving myocardial healing, reducing infarct expansion and preventing electrical instability (10–14). Although this hypothesis has been investigated for so-called "delayed" (up to 24-h) reperfusion therapy (15,16), only few and conflicting data exist regarding reopening an IRA several days after the acute event (17,18). In view of the experimental (19) and clinical evidence (20,21) that depressed LV function after myocardial infarction (MI) treated by thrombolysis may recover over several weeks and even months (prolonged "stunning"), the question arises as whether it may also be worthwhile to reopen an occluded IRA several days or weeks after the acute event in asymptomatic patients. We postulated that such recanalization might reduce LV dilation, delayed LV remodeling and thus preserve LV function, especially if the infarcted myocardium was large and reopening could be accomplished within a certain time window after abrupt occlusion.

Patients. The study included 16 selected patients (12 men, 4 women; mean ± SD age 55 ± 11 years) with a first Q wave anterior wall MI, without a history or evidence of a previous MI and without postinfarction angina. All 16 patients had undergone left heart catheterization within 2 weeks of the acute event and fulfilled the following criteria: proximal left anterior descending coronary artery (LAD) occlusion with no or only minimal anterograde blood flow, Thrombolysis in Myocardial Infarction (TIMI) flow grade 0 or 1, infarct zone dys/akinesia or severe hypokinesia and no additional major epicardial coronary artery stenosis >75%. The patients had no

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Manuscript received November 6, 1997; revised manuscript received March 4, 1998, accepted March 16, 1998.

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0735-1097/98/$19.00
PII S0735-1097(98)00188-0

Abbreviations and Acronyms

CT = computed tomography
IRA = infarct-related coronary artery
LAD = left anterior descending coronary artery
LV = left ventricular
LVEF = left ventricular ejection fraction
MI = myocardial infarction
MRI = magnetic resonance imaging
TIMI = Thrombolysis in Myocardial Infarction

chest pain while climbing flight of stairs but had no formal symptom-limited exercise test before angiography. After providing written informed consent, all patients were randomized to immediate (n = 8) or delayed (after 3 months, n = 8) percutaneous transluminal coronary angioplasty (PTCA) to reopen the IRA.

Methods. A consecutive series of patients who fulfilled the aforementioned clinical and catheterization criteria were asked to participate. After providing written informed consent, all patients underwent cardiac magnetic resonance imaging (MRI). Patients randomized to immediate PTCA underwent this intervention within 17 days of the acute event. All patients were then discharged with a recommended medical treatment, including warfarin or aspirin, an angiotensin-converting enzyme inhibitor at a maximally tolerated dose and other medications as needed. After 3 months, all patients reported to the hospital for clinical examination and repeat cardiac MRI and full left heart catheterization. Patients randomized to delayed intervention underwent initial PTCA. Patients randomized to immediate PTCA who had significant restenoses underwent repeat PTCA. A final investigation on an outpatient basis was performed after 1 year and included clinical examination, exercise testing and cardiac MRI. The study protocol was approved by the ethics committee of the University Hospital of Basel, Switzerland.

Left heart catheterization. Left heart catheterization was performed according to the standard Judkins technique with biplane (30° right anterior/60° left anterior projections) ventriculograms in all patients at baseline and after 3 months. Left and right coronary arteries were filmed in multiple projections after administration of sublingual nitroglycerin. Coronary blood flow was visually assessed according to TIMI criteria (22). Collateral circulation to the occluded LAD was classified as follows: 0 = absent; 1 = minimally visible but no filling of the epicardial vessel; 2 = moderately visible with some, but incomplete, filling of the epicardial vessel; and 3 = full collateralization with retrograde filling of most of the epicardial LAD. Biplane LV volumes and LV ejection fraction (LVEF) were determined by the area–length method by a technician blinded to the study and time after infarction (23). Wall motion analysis was performed using the centerline method (24): Characterization of the infarct zone included percent consecutive abnormal chords >1 SD below the norm (percent abnormal chords) and the mean excursion of the most abnormal 50% of chords in the infarct region (expressed as the number of standard deviations/chord).

MRI. *Image acquisition.* Cine MRI of the left ventricle was performed with a commercially available 1.5-T superconducting magnet (Siemens Magnetom). Electrocardiographic triggered fast gradient echocardiographic images with an echo time of 14 ms and a flip angle of 30° were applied. Two levels with a slice thickness of 8 mm were scanned simultaneously, resulting in 12 to 16 frames/heart cycle for each slice, depending on the patient's heart rate. A multislice coronal, axial and parasaggital localizing sequence was used to define the angulation of the LV long axis relative to the x- and z-plane. The left ventricle was then covered from the apex to the base in a short-axis view (perpendicular to the LV long axis) with 8 to 12 slices 8 mm thick (25). Additional slices were acquired in a four-chamber view (parallel to the LV long axis).

Image analysis. For analysis, cine frames showing the maximal and minimal LV cross-sectional areas and, where possible, the opening or closing of the mitral valve were used to define end-diastole and end-systole. LV volumes were calculated as the sum of the cavity area times slice thickness of all slices covering the left ventricle. Myocardial mass was calculated as the sum of the myocardial area times slice thickness of all slices covering the entire left ventricle times specific myocardial gravity (1.05). All analyses were performed independent of and blinded to the study protocol.

Coronary angioplasty. PTCA for recanalization of occluded coronary arteries was performed using an extrastiff guide wire (26). During the procedure, patients were treated with heparin up to an activated clotting time ≥300 s, and heparin was continued for 16 to 20 h after the procedure. A coronary stent was placed successfully in one patient to stabilize a major dissection. Repeat PTCA for restenosis was performed according to standard over the wire techniques.

Statistics. Results are presented as mean value ± SD. Analyses were performed according to the intention to treat principle. The local ethics committee requested a preplanned interim analysis after enrollment of 16 patients. Comparisons between groups were done with the Student *t* test for unpaired samples and the chi-square test where appropriate. For comparisons within one group over time, a two-way repeated measures analysis of variance was applied, followed by a Student-Newman-Kents post hoc test. Significant differences were assumed for $p < 0.05$.

Results

Baseline characteristics (Table 1). The two patient groups were well matched for all clinical and catheterization findings at baseline. Overall, 63% of patients had received thrombolysis during acute MI, and catheterization was performed 10 ± 4 days (range 7 to 17) thereafter.

Angioplasty success. In the immediate angioplasty group, the occluded LAD could be reopened, and full anterograde flow was restored in all eight patients; however, 50% to 75% residual stenoses remained in two. After 3 months, significant

Table 1. Comparison of Baseline Characteristics*

	Immediate PTCA (n = 8)	Delayed PTCA (n = 8)
Mean age (yr)	58	51
Range	43–72	36–67
Men	5	7
Thrombolysis	5	5
Time to cath (days)	10 ± 3	9 ± 3
LAD occlusion: prox/distal	2/6	3/5
2nd vessel with 50–75% stenosis	1	2
Visible collateral channels, grade	1.3 ± 0.4	1.4 ± 0.7
EDVI (ml/m^2)	130 ± 76	114 ± 28
ESVI (ml/m^2)	61 ± 34	57 ± 15
LVEF (%)	52 ± 4	49 ± 5
Anterior wall motion (SD/chord)	−2.4 ± 0.9	−2.8 ± 0.6
Abnormal chords (%)	52 ± 16	62 ± 17

*p = NS for all comparisons. Data presented are mean value ± SD or number of patients, unless otherwise indicated. cath = catheterization; EDVI = end-diastolic volume index; ESVI = end-systolic volume index; LAD = left anterior descending coronary artery; LVEF = left ventricular ejection fraction; prox/distal = proximal/distal to first septal side branch; PTCA = percutaneous transluminal coronary angioplasty.

restenosis (≥75% lumen diameter) occurred in four patients (50%), of whom repeat PTCA was successful in three but was not attempted in one patient with reocclusion and an initial suboptimal PTCA result. In the delayed PTCA group, the IRA was still totally occluded in five patients, showed a high grade stenosis in two and a 60% stenosis in one. PTCA was successful in six of six patients, was not attempted in the patient with the 60% residual stenosis and was not possible (no vessel stump visible) in one patient.

Clinical outcome/follow-up. Three of the four patients in the immediate PTCA group with restenosis reported angina pectoris (New York Heart Association functional class III or IV) at the 3-month follow-up visit (92 ± 8 days after the acute event), one of whom had been admitted after 6 weeks for severe unstable angina. Because of urgent repeat PTCA in this patient, neither control ventriculography nor noninvasive testing was done. In contrast, only one patient without immediate intervention but with a 70% left marginal coronary artery stenosis complained of mild angina (functional class II) after 3 months; in addition, two patients in this group reported shortness of breath. All patients were alive at 1 year, and none complained of anginal chest pain. One patient in the immediate and two in the delayed PTCA group reported dyspnea. Medical therapy was comparable in both groups and included aspirin or warfarin in all patients and angiotensin converting-enzyme inhibitors in five immediate PTCA group and six delayed PTCA group patients (p = NS). Heart rate at rest was significantly higher in patients with delayed PTCA (80 ± 8 vs. 63 ± 8 beats/min, $p < 0.01$), but ergometric results and maximal rate–pressure product achieved were not significantly different between the two treatment groups.

Effects of immediate PTCA on hemodynamic variables. The changes in LV volumes and LVEF from the baseline to the 3-month follow-up contrast ventriculogram for both groups are shown in Figure 1. There were no significant changes in these measurements for patients with delayed PTCA, in marked contrast to the significant improvement in LV end-systolic volumes and LVEF observed in patients with immediate PTCA ($p < 0.01$ each). This amelioration was clearly due to improved regional wall motion, as shown in Figure 1. Measurements of regional function in the infarct zone were significantly better in this group compared with baseline measurements: percent abnormal chords improved from 57.0 ± 22.0% to 27.7 ± 27.5% ($p < 0.01$) and the mean excursion of the most abnormal 50% of chords from −2.4 ± 0.9 to −1.0 ± 1.9 SD/chord ($p < 0.06$). In contrast, in patients with delayed PTCA, these measurements did not change significantly over the first 3 months after infarction.

Effect of delayed PTCA on 1-year hemodynamic variables. The changes in LV volumes and LVEF as determined by cardiac MRI from baseline up to 3 months and 1 year are shown in Figure 2. During the first 3 months, the noninvasive variables of LV function paralleled those determined by

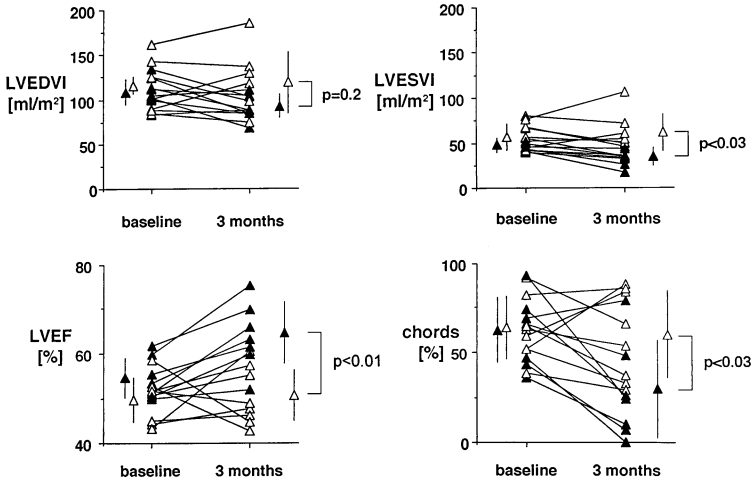

Figure 1. LV volumes, LVEF and percent abnormal chords determined by biplane contrast ventriculograms at baseline and after 3 months in patients with immediate (**solid triangles**) or delayed (**open triangles**) PTCA. LVEDVI = left ventricular end-diastolic volume index; LVESVI = left ventricular end-systolic volume index, chords = percent abnormal chords (variable of infarct zone wall motion; see Methods).

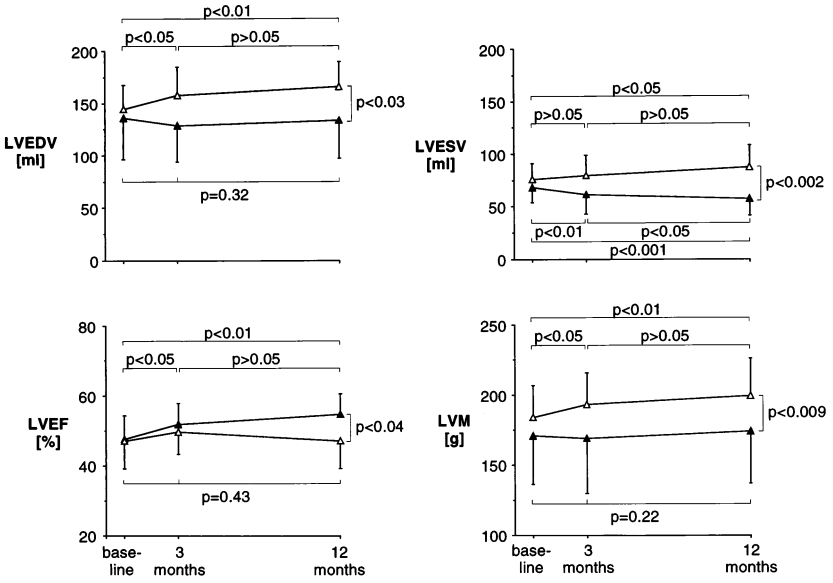

Figure 2. LV volumes, LVEF and mass as determined by MRI at baseline, after 3 months and at 1 year in patients with immediate (**solid triangles**) and delayed (**open triangles**) PTCA. LVEDV = left ventricular end-diastolic volume; LVESV = left ventricular end-systolic volume; LVM = left ventricular myocardial mass.

contrast ventriculography, and the differences between the two patient groups tended to increase up to the 1-year follow-up visit. Thus, the changes in LV volumes and LVEF over the 1-year period were significantly different and pointed in opposite directions for patients with delayed versus those with immediate PTCA (Table 2). Therefore, the improvement seen during the first 3 months after successful immediate PTCA, (i.e., reduced LV end-systolic volumes without increased end-diastolic volumes) was maintained over the 1-year period, whereas delayed PTCA could not prevent LV dilation and increased LV mass and thus functional deterioration (Fig. 2). In the three patients with spontaneous reopening of the IRA between 3 weeks and 3 months, the changes in LVEF over time were variable: One showed continued improvement; one showed deterioration; and one showed initial improvement, followed by a later decrease.

Discussion

The most important result of the present pilot study—to our knowledge the first prospective, randomized investigation on the effect of late reopening of an occluded IRA on LV function—is the demonstration of a significant benefit when an occluded IRA is reopened within 2 to 3 weeks after MI on LV function and remodeling in selected patients, indicating the presence of viable myocardium despite Q waves, infarct zone akinesia and absence of symptoms. This effect was due to improved regional infarct zone wall motion and a decrease in LV end-systolic volumes and suggests a prolonged state of "hibernation" in patients, followed, after restoration of flow, by "stunning," with partial recovery of LV function. This effect was no longer seen when the occluded IRA was opened only after 3 months. In contrast, the left ventricle in these patients showed progressive dilation and remodeling. There was a high initial PTCA success rate early after MI but a high restenosis rate as well. Restenosis in these patients was accompanied by new severe angina as a clinical indicator of salvaged myocardium, which did not occur after delayed PTCA. Thus, there seems to be a short time window after the acute event during which it might still be worthwhile to reopen an occluded IRA, even in asymptomatic patients.

Table 2. Changes in Left Ventricular Volumes, Mass and Ejection Fraction Between Baseline and 1 Year Determined by Cardiac Magnetic Resonance Imaging in Both Groups of Patients

	Immediate PTCA (n = 8)	Delayed PTCA (n = 8)	p Value
ΔEDV (ml)	−2.8 ± 6.9	+21.2 ± 17.3	0.007
ΔESV (ml)	−11.6 ± 3.3	+11.8 ± 11.6	0.0005
ΔLVEF (%)	+8.0 ± 4.3	0.0 ± 6.3	0.02
ΔLVM (g)	+3.0 ± 6.6	+15.6 ± 11.7	0.02

Data presented are mean value ± SD. EDV = end-diastolic volume; EDS = end-systolic volume; LVM = left ventricular mass; Δ = change in; other abbreviations as in Table 1.

PTCA success in total coronary occlusions. Duration of occlusion is a key factor for success of PTCA for total occlusions (27), as noted in the present study. Our results compare favorably with three recent reports on delayed reopening of occluded coronary arteries 1 to 21 days after the acute event, with primary success rates of 72% (18), 81% (15) and 93% (17). The restenosis rate after PTCA for total occlusion is high (27); however, as in the present study, in the majority of patients recurrences were caused by restenoses rather than reocclusion. It remains to be shown whether regular use of coronary stenting may also significantly reduce the recurrence rate in these patients (28). In the present study, three of four patients in the immediate PTCA group with restenoses complained of significant chest pain. In this setting, chest pain is an important clinical marker of preserved viability

and is even more important if a similar recurrence rate is assumed for patients in the delayed PTCA group, who reported no such symptoms.

Effect on LV function and remodeling. In the Thrombolysis and Angioplasty in Myocardial Infarction-6 (TAMI) study (15), 71 patients with occluded coronary arteries 24 h after failed thrombolysis were randomized to PTCA or no PTCA. Although there was improved LV function at 1 month, no advantage could be demonstrated after 6 months. However, only 50% of patients could be restudied, with patency rates of 89% for immediate PTCA and 60% for delayed PTCA. In addition, only 50% of patients had a large anterior infarction, which would have shown a potential benefit more clearly. Similarly, Dzavik et al. (18) found a significantly greater improvement in patients with a patent IRA at follow-up than in those with an occluded artery. Patients with the lowest LVEF and those with an anterior infarction had the greatest improvement. Because gated radionuclide ventriculograms were used in most patients for follow-up evaluation, no detailed function analysis was possible.

In a recent nonrandomized study of similar patients, Pizzetti et al. (29) also described a progressive improvement in echocardiographically determined LV function and volumes in 16 patients with successful reopening of an occluded LAD within 18 days of MI, in contrast to further deterioration of these variables in 11 patients with failed PTCA. Because that study was not randomized, other factors than vessel patency may have influenced the result. Furthermore, determination of LV volumes by echocardiography is heavily dependent on geometric assumptions, which usually are not met after a large Q wave MI, in contrast to biplane contrast ventriculograms and MRI studies of the heart, which were used in the present investigation. The findings of both studies suggest that there is still viable myocardium despite prolonged (1 to 3 weeks) occlusion of the IRA. However, viable myocardium is present only for a short period of time after the acute event. In the present study it could no longer be detected in patients with an IRA that was reopened after 3 months. In these patients the effect of PTCA on LV function was as insignificant as that reported in other patients with chronic total occlusions (30,31). Thus, there seems to be a short time window of only 2 to 3 weeks in which myocardium may still be salvaged and remodeling of the LV prevented. The presence of collateral channels will most likely prolong this time window by inducing a state of so-called "hibernating" myocardium (32,33). That three of eight patients without early PTCA had an open IRA after 3 months (i.e., that these vessels spontaneously reopened somewhere between 1 and 3 months) is in agreement with the overall findings and strongly supports the notion that this time window is short (i.e., 2 to 3 weeks rather than 2 to 3 months). Because we do not know the exact point in time at which these arteries reopened, we can only speculate that it might have been earlier in the patient with continued improvement of LV function than in the other two.

Limitations of the study. This was a small pilot study of patients highly selected for the largest possible benefit—large infarct area, first Q wave MI—and minimal confounding effects—no multivessel involvement, no previous infarction. Thus, the study group represented <3% of all patients with an MI and may serve only to prove a hypothesis; the results may not be translatable to all post-MI patients. Much larger studies will be necessary to define the relevance of the present findings to other patients, other IRAs, other factors involved and, most important, whether this intervention will also improve survival. In view of the published reports, the present findings suggest that a certain large area at risk is necessary for a positive effect to be noticeable and that the time window for opening the IRA appears to be limited. Further studies will be necessary to assess the role of noninvasive markers of myocardial viability to define this time window more clearly.

Conclusions and implications. The findings of the present pilot study suggest that there is a prolonged state of "hibernating" myocardium after MI in humans and that restoration of anterograde flow within 3 weeks of the acute event may promote recovery of LV function and prevent LV dilation. However, this assumption may only be true in patients with a large anterior infarction, as studied here, and only if the artery is reopened during a short time window of 2 to 3 weeks after the acute event; the effect of later interventions may no longer be beneficial. The present pilot study may therefore serve as basis for a large prospective mortality trial on this important issue.

We thank Franziska Bader, MTRA for technical assistance.

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